

PARVOVIRAL ENTERITIS IN A DOG: CASE REPORT AND REVIEW OF THE LITERATURE

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ABSTRACT

Canine parvovirus enteritis is a highly contagious serious disease of young dogs under the age of 6- 20 weeks. Infected puppies shed virus in their faeces thereby contaminating the environment and increasing the chances of infection of naïve puppies. All puppies are at one point at great risk of infection even after vaccination because of period of “window of susceptibility”. Therefore, dog owners should strictly observe good hygiene and disinfection of their environment using bleach at 1: 32 dilution and adequate exposure of contaminated fomites to sunlight and heat to destroy the virus. This case in a 6months Alsatian male dog recorded frank foul smelly bloody diarrhoea, drooling salivation, emaciation, hyperesthesia, anorexia, depression and severe vomition. This report therefore reviewed parvovirus enteritis disease in dogs with a view to highlight the trend of the disease and ways of management.

KEYWORDS: Dogs, parvovirus, enteritis.

INTRODUCTION

Canine parvovirus infection was first recognized in 1978 and subsequently disease spread worldwide (Carmichael 2005). Infection in dogs is mainly seen in young puppies between 6-20 weeks of age (Mercks 2006) and some adult dogs. The disease is caused by 2 strains of virus namely CPV2a and CPV2b identified in 1978 and 1984 respectively (Shackelton et al., 2005). A newer strain, CPV2c have recently emerged with its associated danger because of the difficulty in its isolation in the laboratory (Foster and Smith 2011). Dogs can get infection through direct contact with contaminated faeces (Prittie 2004; Animal hosp. 2011) and indirectly by contact with contaminated fomites such as clothings, hospital benches and tables, food pans and kennel floors (Foster and Smith 2011; Wikipedia 2011). Indirect transmission is due to the hardy nature of the virus to adverse environmental condition and can remain infectious in contaminated environment for months (Animal hosp. 2011). Infection can also be through insect and rodent vectors (Foster and Smith 2011). The disease manifests in two forms, the cardiac and the intestinal form in puppies and is very severe in those with no maternal antibodies and vaccination history (Wikipedia 2011). The cardiac form is only seen in young puppies infected in utero manifests with respiratory or cardiovascular failure (Schatzberg et al., 2002; Foster and Smith, 2011; Wikipedia 2011). However, this form of the disease no longer exists because of maternal immunity (Animal hosp. 2011; Mercks 2006). Canine parvoviral enteritis is a highly contagious and severe viral form of the disease (Debram 2011; eHow 2011; Mercks 2006). Virus has affinity and invades rapidly mitotic cells such as is seen in the intestine, bone marrow and lymphnodes after an incubation period of 7-14 days causing intestinal impairment (Foster and Smith 2011). Invasion of the bone marrow causes a decrease in the white blood cell count leading to increased susceptibility to bacterial infections and sometimes shock due to endotoxaemia (Debram 2011). Virus may be found in the faeces days before appearance of clinical signs and may continue to the next 2 weeks (Foster and Smith 2011). Clinical signs includes: severe vomition, diarrhoea, dehydration, foul smelly dark bloody faeces, anorexia, nausea, depression and in very acute case death occurring 2 days post infection (eHow 2011; Foster and Smith 2011). Breeds such as Doberman pinschers, Rottweilers, German shepherds, Staffordshire terriers, black Labrador retrievers and Dachshunds are particularly susceptible to the disease (Nelson et al., 1998; Debram 2011; Foster and Smith 2011). The commonest diagnostic technique of parvovirus enteritis is by CITE test which is an enzyme linked immunosorbent assay antigen test on the faecal sample within 20 minutes, a thorough physical examination, laboratory test on the complete blood count (CBC) and chemistry profile test to ascertain the severity of the disease (foster and smith 2011). X-ray test can be done to reveal swollen intestinal loop containing gas and fluid characteristic of parvoviral enteritis and PCR technique to detect the viral antigen (Jones 1997; Foster and Smith 2011). Treatment is mainly a vigorous supportive management of the infected puppy by effectively and adequately replacing lost fluid using a poly ionic isotonic dextrose solution given intravenously, subcutaneously or orally in less severe cases (Foster and Smith 2011).

Blood or plasma transfusion may be given as the case may be along with broad spectrum antibiotics such as Cefoxin, metronidazole, timentin or enrofloxacin with adequate amount of vitamin B complex and iron dextran to prevent endotoxaemia (Ettinger et al., 1995; Foster and Smith 2011). Food and water should be discontinued in cases of persistent vomiting, diarrhoea and replaced with fluid and commercial nutritional diets that are less complex in digestion and less irritating to the intestinal mucosa such as Hills[®], Prescription diet[®], canine 1/d[®] and feline 1/d[®]. The dog's normal food should be gradually replaced after recovery from the enteritis (Ishiwata et al., 1998). Infected puppies may also be treated with interferon omega (rFelfn-w t the dose of 2.5mu/kg for 3 consecutive days to reduce mortality and improvement of clinical signs (Kuwabara 2006; Martin et al., 2002; Virginie et al., 2002; De Mari et al., 2003; Ettinger et al., 1995). However, once an infected dog recovers from the disease, it becomes immune for life. The main stay of Prevention is vaccination of puppies with modified live vaccines at 6-8 weeks of age, repeated at every 3 weeks until the puppy is 16-20 weeks of age, then a booster dose at one year and every 1-3 years thereafter (Foster and Smith, 2011). This vaccination protocol was adopted because of the fact that maternal antibodies interfere with immune vaccine response and also at one point in time puppies have some level of maternal antibodies which are not capable of protecting them against the disease but sufficient enough to prevent their immune vaccine response, thereby leaving the puppy unprotected and at great risk of infection. This period in a puppy is known as the "window of susceptibility" and therefore the vaccination protocol is designed to provide protection at all times (Foster and Smith 2011). There should also be a strict hygiene programme of disinfecting infected fomites with sodium hypochlorite (bleach solution at 1: 32 dilution) (Macintire 2004). The bleach may be impaired by the presence of inorganic matter therefore surfaces should be first cleaned with commercial disinfectants before the use of the bleach. Prognosis in parvovirus enteritis is favourable in aggressive therapy but grave in untreated cases (Prittie 2004).

Case report

A black and brown 6 months Alsatian male dog weighing 12kg was presented to the veterinary clinic on 12th march, 2011. The client complained of inappetence in the dog for the past ten days, groans all the night, hypersensitive to touch, vomiting, passes loose brown faeces but not diarrhoeic and had no previous vaccination of any sort. Thorough physical and clinical examination revealed heart rate of 135/min, pulse rate 130/min, respiratory rate 44/min, temperature 40.7^oC, poor hair coat, presence of ticks and fleas, respiratory rales, drooling salivation and dullness. On digital rectal investigation, there was a sudden discharge with great pressure of frank foul smelly bloody diarrhoea. A tentative diagnosis of parvovirus enteritis, corona virus, hookworm infection and coccidiosis was made and confirmed by elimination. Analysis of the faecal sample was negative for helminths ova. Confirmative diagnosis was made on a positive faecal Eliza test for parvo and on the characteristic foul smell. Treatment was managemental by giving oxytetracycline 200mg i/m, iron dextran 100mg i/m, vitamin B complex 150mg i/m, multivitamine 2-5ml/kg i/m, and 5% dextrose 0.9% saline given orally and subcutaneously. The treatment lasted for a week before the gradual recovery of the dog.

DISCUSSION/CONCLUSION

The case reported parvoviral enteritis in a dog of 6 months of age. This is in line with reports of (Merck 2006; Debram 2011 and eHow 2011) as they said that disease is associated with dogs within 6-20 weeks of age. Puppies are known to be highly susceptible to this disease because of period of window of susceptibility during which the low level of maternal antibody in the puppy is not able to protect it from the disease and will also prevent vaccine response thereby leaving the puppy at a vulnerable condition and at great risk of infection (Foster and Smith 2011). Adult dogs are generally resistant because of previous exposure to the virus; immunity to parvoviral enteritis is known to be life long except in immunosuppressed dogs. The case was reported in an Alsatian dog which is in agreement with the observation of (Debram 2011; Foster and Smith 2011; Nelson et al., 1998).

The clinical signs observed are in line with the classical signs of parvoviral enteritis (Debram 2011; eHow 2011; Foster and Smith 2011). The idiosyncrasy of this report is the projectile frank blood diarrhoea observed in this on rectal examination. This could be as result of additional irritation to the already attacked intestinal epithelium by the examiner's finger. The spurting out of the blood was with a great pressure that splashed on the examiner's lab coat. The respiratory rales and increased heart rate and pulse rate is a manifestation of the cardiac form of the disease however of less severe. This is contrary to the general belief of non existence of this form of parvo because of transfer of maternal antibodies (Animal Hospital USA, 2011). We think that the disease is still in existence however mild because of the presence of maternal antibodies which is dependent on the immune status of the bitch, its ability to produce adequate protective antibodies titre transferable to its foetus. Therefore, immunocompromised bitches or those under corticosteroid therapy can whelp puppies that may show cardiac

form of the disease and some of the puppy may have congenital cerebral hypoplasia (Schatzberg et al., 2002). So far there has not been a recorded case of drooling saliva in parvoviral enteritis. However, it was observed in this case and perhaps may be due to fear and excitement over been examined by clinicians. The systematic approach in the elimination of the differential diagnosis is based on the fact that vomition seen in this case is not observed in coronavirus infection in dogs. Laboratory investigation of the faeces shows no evidence of hookworm infection. Coccidiosis is an option that should be considered however as opportunistic infection. It is known that on when parvovirus destructs the intestinal epithelium, it creates a conducive environment for bacterial and protozoans colonization leading to secondary bacterial infection complicating the bloody diarrhoea seen in this case. The treatment was only managerial by administering oxytetracycline 200mg, to cheekmate endotoxaemia and secondary bacterial complications. However, Macinitire (2004) prefers the use of enrofloxacin, trimethoprim, metronidazole and cefoxitin.

Iron dextran 100mg was given as a haematinic to boost replacement of blood loss.

B complex is a good immune booster for quick recovery of the dog. The most important of all is the fluid therapy of 5%dextrose 0.9%saline given to replace lost fluid and electrolytes. This is in line with Foster and Smith (2011) who said that recovery of infected puppy is dependent on the effective and adequate fluid replacement. However, the use of lactated ringers' solution would have been better in replacing lost electrolytes. Importantly, this report did not administer any intestinal protectants and astringents which would have help reduce the scouring and the dehydration status of the dog. Conclusion, Parvoviral enteritis is an also endemic disease in our environment because of its hardy nature and also newer strains of high virulence ii emerging. Therefore, dog owners and veterinarian should ensure that precious dogs are vaccinated following the vaccine protocol and ensuring good hygiene using bleach.



Fig 1, Projectile bloody diarrhoea provoked in an infected puppy with parvoviral enteritis on rectal palpation on the examination table.



Fig 2. Notice the drooling saliva in the case of parvoviral enteritis.



Fig.3. Showing emaciation in the dog typified by prominent rib cage bones sequel to parvoviral enteritis.

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